# The Transient and Permanent Effects of Alcohol Consumption Upon Cytokine Sensitivity in Nerve Fibers – Implications for Treating Hypertensive Disorders

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## Introduction

Alcohol consumption is associated with a series of harmful effects, some of which are better-understood than others. This paper will address itself to the effect of edema and the parallel effect of blood vessel constriction including pulmonary hypertension even in the absence of markedly increased cytokine levels.

## Abstract

In response to the condition of hypothermia, the body releases cytokines in order to restrict blood flow to the extremities in order to protect vital organs. Alcohol consumption, both in the short-term and in association with chronic exposure to the substance, ostensibly results in both edema and increased cytokine sensitivity in select parts of the body. This is to say that although alcohol use is associated with general hypertension, it has a specific association with pulmonary hypertension and myocarditis. Thus, some nerve groups are affected to a greater degree than others.

Importantly, people who consume alcohol report, “feeling warm.” There is a widespread misconception that decreased body temperature produces this sensation and that it is somehow the inverse of the effect in which a person with a fever feels cold. Alcohol does not cause body temperature to decrease, however, people experiencing hypothermia are advised to avoid alcohol not because it will make them colder, but because it will inhibit circulation and cause them to potentially fall asleep whereas they need to remain physically active to counteract the effects of hypothermia.

That said, medical professionals do not have an adequate medical explanation for why it is that people feel warm after consuming alcohol, but an understanding of this phenomenon could provide insights into what is really transpiring when a person experiences poor circulation; either related to alcohol or not.

This author published a paper on the topic of the physical mechanisms which underpin sensations generated by nerve fibers including sensitivity to heat, sensitivity to cold, sensitivity to pressure, et cetera. Pain signals are, according to that paper, related to the release of chemicals from nerve fibers which, when present around the exterior of the fibers, produce a modified return signal which is interpreted as pain. This is part of a somewhat unorthodox, currently unaccepted model of the human nervous system. The currently accepted model states that nerves generate electrical signals in response to stimuli which are interpreted by the brain. This author’s model, notably, is based upon the idea that the brain is constantly sending “ping”

signals to various tissues and measuring the quality of the return data to make inferences about the environment.

If the presence of alcohol causes a person to “feel warmer,” this may be due to the fact that alcohol, when it interacts with the exterior of nerve fibers, prompts the release of chemicals usually associated with elevated temperature. If the nervous system believes itself to be warmer than it really is, cytokines would be produced in lesser amounts in response to this. Eventually, the chemical responsible for producing this artificial sensation of warmth is exhausted. Although the hypothalamus is well-aware that body temperature is normal under such a circumstance, the nerves fibers believe that the body is cold. Repeated exposure to alcohol could then, therefore, be predicted to cause the nerve fibers to fail to produce adequate quantities of the “warm enough” chemical signal, causing the nerves to believe that conditions are actually colder than they are. In short, it would create a conflict between the brain’s system for regulating body temperature and the nervous system’s mechanism for doing this (cytokines are a micro-cellular tertiary adjunct to this system, meaning that the human body has at least three distinct means of assessing and managing its own temperature autonomically.) In response, sensitivity to cytokines is elevated through epigenetic switching. The hypothalamus, under this circumstance, insists that the body is “warm enough” but the nerve fibers beg to differ and change their own mode of operation, creating a conflict in which a person’s blood vessels constrict inappropriately under normal conditions.

When a patient takes a drug designed to reduce localized hypertension, the mode of effication may actually be a desensitization to cytokines brought on by blocking the cytokine receptors in those nerve fibers, although the mode of effication of those drugs is not currently understood by doctors. It would make sense for doctors to learn something about the mode of effication of the drugs they prescribe if they wish to cure disease.

## Conclusion

A more permanent treatment of such conditions would entail permanent normalization of cytokine sensitivity in the target nerve fibers through therapies which throw the epigenetic switches in the nerve tissues back to their default positions. This should make it possible to cure hypertensive disorders in a single treatment; preferable to relying upon the use of receptor blockers on an ongoing basis.